

Internal Medicine Sub-internship Training Problem #6: Arrhythmias

T. Robert Vu, M.D.
William J. Gill, M.D.
Indiana University School of Medicine

Note: The correct multiple choice answer for each question is underlined.

Specific Learning Objectives:

The knowledge, skills, and attitudes towards recognition/diagnosis and management of common arrhythmias are noted in the CDIM Internal Medicine Sub-internship Curriculum.

➤ Hyperkalemia-induced EKG changes

1. A 48-year-old man presented to the emergency department complaining of severe 10/10 chest pain. He has end-stage renal disease and receives hemodialysis three days a week. He just used cocaine about 4 hours ago. His current EKG and a comparison tracing from two days prior are shown. Which of the following is the most appropriate immediate treatment for him?
 - a. Intravenous thrombolytics
 - b. Emergent left heart catheterization with coronary angiography
 - c. Aspirin, intravenous heparin, clopidogrel, and intravenous glycoprotein IIb/IIIa inhibitor (e.g., eptifibatide)
 - d. Calcium gluconate, intravenously

Explanation to above answer: This EKG (when compared to a baseline tracing done two days prior) showed tall, peaked, and symmetrical T waves and widened QRS duration due to hyperkalemia. Although in the setting of severe chest pain and recent cocaine use, this EKG could possibly be interpreted as an acute myocardial infarction with hyperacute T waves preceding the ST segment elevation. However, there are no “tombstoning” of the ST segments seen on this tracing to indicate a myocardial injury pattern. Additionally, hyperkalemia should always be suspected in a patient with end-stage renal disease who presents with abnormal and suspicious repolarization changes on EKG. Finally, administration of intravenous calcium gluconate will rapidly bring the hyperkalemia-induced repolarization changes on EKG back to baseline to allow a rapid confirmation of the suspected diagnosis.

2. Corollary questions to the above case #1:
 - a). What other diagnostic test would you need to confirm your suspicion?
Answer: serum electrolytes, focusing on serum potassium and bicarb levels. This particular patient’s serum potassium level turned out to be 7.3.

- b). Describe, in progressive stages, the EKG changes one would see with untreated, severe hyperkalemia. Are any seen here on this tracing?

Answer:

- i) Tall, peaked, symmetrical T waves (seen on this tracing), then...
 - ii) PR interval and QRS duration both lengthen (the latter is seen on this tracing), then...
 - iii) P wave disappears (not seen), then finally...
 - iv) QRS further widens into “sine wave” pattern (not seen)
- c). What is calcium’s mechanism of action in this particular case?
- Answer: calcium protects the myocardium by antagonizing the hyperkalemia-induced depolarization of the resting membrane potential.
- d). What are some of the other useful adjunctive medical therapies for this particular case and their mechanisms of action?
- Answer:
- i) Insulin & glucose (D50) via intravenous injection, or inhaled beta-2 adrenergic agonist. Both therapeutic measures drive potassium into the cells by increasing Na-K-ATPase activity.
 - ii) Sodium polystyrene sulfonate (Kayexalate) acts in the gut as a cation exchange resin, taking up potassium and releasing sodium. *Caution:* This agent is commonly used in combination with sorbitol to enhance fecal elimination; however, a number of cases of colonic necrosis and perforation associated with the administration of these 2 agents have been reported. This colonic risk appears to be highest when these 2 agents are given to post-operative patients.
 - iii) Sodium bicarbonate raises the systemic pH and causes hydrogen ion release from the cells in exchange for potassium movement into the cells to maintain serum electroneutrality.
- e). What should be the ultimate therapeutic management plan for this patient’s current problem, especially if the above therapeutic measures are unsuccessful?

Answer: acute hemodialysis.

References:

Slovic C, Jenkins R. ABC of clinical electrocardiography: Conditions not primarily affecting the heart.[erratum appears in *BMJ* 2002 Aug 3;325(7358):259]. *BMJ*. 324(7349):1320-3, 2002 Jun.

Gennari FJ. Disorders of potassium homeostasis. Hypokalemia and hyperkalemia. *Critical Care Clinics*. 18(2):273-88, vi, 2002 Apr.

Mahoney BA, Smith WA, Lo DS, Tsoi K, Tonelli M, Clase CM. Emergency interventions for hyperkalaemia. *Cochrane Database of Systematic Reviews*. (2):CD003235, 2005.

Sood MM, Sood AR, Richardson R. Emergency management and commonly encountered outpatient scenarios in patients with hyperkalemia. *Mayo Clinic Proceedings*. 82(12):1553-61, 2007 Dec.

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➤ Paroxysmal supraventricular tachycardia

3. A 22-year-old man without any known coronary artery disease presents with palpitations and anxiety. His blood pressure is 120/72, and he appears relatively comfortable. Which of the following is the most appropriate next step in his care?
 - a. No therapy needed other than reassurance
 - b. Direct current (DC) cardioversion after adequate sedation is achieved
 - c. Digoxin, intravenously
 - d. Carotid sinus massage or Valsalva maneuvers

4. Corollary questions to the above case #3:
 - a). What is the rhythm to question #3 above?
Answer: Paroxysmal supraventricular tachycardia.
 - b). What is the mechanism of this arrhythmia?
Answer: AV nodal re-entry. A big hint on this tracing is the presence of retrograde P waves indicating impulse conduction up the fast pathway within the AV node in a retrograde fashion.
 - c). If the above therapeutic measure is unsuccessful, what is the next appropriate management step?
Answer: adenosine, intravenously.

References:

Esberger D. Jones S. Morris F. ABC of clinical electrocardiography. Junctional tachycardias. *BMJ*. 324(7338):662-5, 2002 Mar 16.

ACC/AHA/ESC Guidelines for the Management of Patients With Supraventricular Arrhythmias—Executive Summary. *Journal of the American College of Cardiology*. 42(8):1493-531, 2003 Oct 15.

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➤ Atrial arrhythmias

5. A 70-year-old man with long-standing hypertension presented with symptoms of congestive heart failure including dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea, and dependent edema. These symptoms have progressively worsened over the past 3 weeks. He has had no chest pain/discomfort, palpitations, cough, or fever/chills. He never smoked. A recent cholesterol level was 158, and he has no known history of diabetes or coronary artery disease. What is the rhythm?
 - a. Sinus tachycardia
 - b. Multifocal atrial tachycardia (MAT)
 - c. Atrial flutter with 3:1 AV conduction
 - d. Atrial flutter with 2:1 AV conduction
 - e. Atrial fibrillation with rapid ventricular response

Explanation to above answer: This tracing shows classic “sawtooth” flutter waves in a 2:1 AV conduction ratio such that the atrial rate is 300 bpm while the ventricular rate is 150 bpm. This rhythm is very regular and, therefore, cannot be MAT or atrial fibrillation.

6. For the patient in question #5 above, which of the following is the most likely cause of his congestive heart failure?
- Viral cardiomyopathy
 - Tachycardia-induced cardiomyopathy
 - Toxin-mediated cardiomyopathy (e.g., alcohol/cocaine/adriamycin)
 - Ischemic cardiomyopathy
7. This electrocardiogram was obtained from a 68-year-old man who presented with dyspnea and palpitations. He has a long history of hypertension and mild congestive heart failure (LV systolic dysfunction). His current blood pressure is 150/90. Physical examination reveals no JVD, an irregularly irregular cardiac rhythm with no audible third heart sound, and clear lungs. All of the following are appropriate management options for this rhythm EXCEPT:
- Digoxin
 - Diltiazem
 - Verapamil
 - Nifedipine
 - Metoprolol

Explanation to above answer: This EKG shows atrial fibrillation with rapid ventricular response in a patient who is hemodynamically stable and is not in overt CHF. All of the listed medications effectively block AV nodal conduction to control heart rate except nifedipine.

8. This rhythm strip was obtained from a 71-year-old man with COPD who presented with severe dyspnea and palpitations. Physical examination revealed labored breathing with bilateral expiratory wheezing and tachycardia. Which of the following is the most appropriate therapy?
- Beta blockade
 - Adenosine, intravenously
 - Beta-2 adrenergic agonist, inhaled
 - Direct current (DC) cardioversion
 - Heparin, intravenously

Explanation to above answer: The rhythm in this case is multifocal atrial tachycardia (MAT). Therapy should be directed towards any underlying condition that may predispose to MAT such as lung or cardiac disease, hypokalemia, and hypomagnesemia. In this particular patient's case, his COPD precipitated the MAT and therapy should be directed at reversing his acute airways obstruction. Heart rate control with either verapamil or beta-blockers should only be considered when the rapid ventricular response worsens myocardial ischemia, heart failure, or oxygenation.

9. Corollary question to above case #8:
- If the rate was less than 100 bpm, what is the rhythm then called?
Answer: wandering atrial pacemaker

References:

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Umana E, Solares CA, Alpert MA. Tachycardia-induced cardiomyopathy. *Am J Med* 2003 Jan;114(1):51-55.

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➤ Drug-induced QT prolongation with resultant torsades de pointes

10. All of the following medications could most likely be responsible for the marked prolongation of the QT interval in this 48 year-old woman EXCEPT:

- a. Azithromycin
- b. Amiodarone
- c. Itraconazole
- d. Calcium
- e. Amitriptyline

Explanation to above answer: This tracing shows a markedly prolonged QT. In fact, there are very prominent U waves on this tracing which should also be counted as part of the QT interval. In general, if the U wave is large (at least 25-50% or more of the height of the T wave), then the measurement of the QT interval should include the U wave. All of the agents listed above, with the exception of calcium, are well-known to prolong the QT interval.

11. This rhythm strip is taken later from the patient in question #10 above. What is the most appropriate next management step?

- a. Amiodarone, intravenously
- b. Lidocaine, intravenously
- c. Precordial thump
- d. No therapy required. This represents artifact.
- e. Magnesium, intravenously

Explanation to above answer: This rhythm strip shows the classic polymorphic VT called torsades de pointes—therapy for which should be intravenous magnesium. Patients with abnormally prolonged QT interval are predisposed to this type of arrhythmia. Other therapeutic alternatives for torsades include isoproterenol and cardiac pacing.

References:

Gupta A, Lawrence AT, Krishnan K, Kavinsky CJ, Trohman RG. Current concepts in the mechanisms and management of drug-induced QT prolongation and torsade de pointes. *American Heart Journal*. 153(6):891-9, 2007 Jun.

Al-Khatib SM. LaPointe NM. Kramer JM. Califf RM. What clinicians should know about the QT interval.[comment][erratum appears in JAMA. 2003 Sep 10;290(10):1318]. *JAMA*. 289(16):2120-7, 2003 Apr 23-30.

Roden DM. A practical approach to torsade de pointes. *Clinical Cardiology*. 20(3):285-90, 1997 Mar.

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➤ Atrioventricular blocks

12. A 66-year-old woman presents to the emergency department with an orbital blow-out fracture following a syncopal episode on her stairs at home. This is her 3rd syncopal episode in the last two weeks. This electrocardiogram was obtained. What is the underlying rhythm?
- Sinus bradycardia with 1st degree atrioventricular block and occasional premature atrial complexes
 - Sinus bradycardia with 2nd degree atrioventricular block, Mobitz type I (Wenckebach)
 - Sinus bradycardia with 2nd degree atrioventricular block, Mobitz type II
 - Third-degree atrioventricular block (complete heart block with AV dissociation)
 - Junctional rhythm
13. Corollary question to above case # 12:
- What is the most appropriate next management step?
Answer: pacemaker insertion
14. A 58-year-old male veteran with a long history of tobacco use as well as hypertension that has been difficult to control. He is now on diltiazem, metoprolol, and hydrochlorothiazide with improvement in his blood pressure which was measured as 144/78 today in your clinic. On cardiac auscultation, you hear dropped beats periodically and so you obtain this electrocardiogram. What is the rhythm?
- Sinus bradycardia with non-conducted PAC's
 - Sinus bradycardia with 2nd degree atrioventricular block, Mobitz type I (Wenckebach)
 - Sinus bradycardia with 2nd degree atrioventricular block, Mobitz type II
 - Third-degree atrioventricular block (complete heart block with AV dissociation)
 - Sinus bradycardia with sinus arrhythmia
15. What's the most appropriate next step in the treatment of this patient in question #14?
- Avoid caffeine and other stimulants
 - Refer to electrophysiologist to urgently place a pacemaker
 - Reduce the dose of his diltiazem and/or metoprolol
 - No treatment necessary; this is sinus arrhythmia which is a normal variant

16. A 60-year-old man presents to your office complaining of feeling excessively fatigued, worsening dyspnea on exertion, and lightheadedness over the past several days. He has a long-standing history of severe ischemic cardiomyopathy. His blood pressure today is 100/60 and has bibasilar crackles on lung auscultation. You obtained this electrocardiogram. Which of the following is the best treatment for his symptoms?
- Dual chamber permanent pacemaker
 - Carvedilol 3.125mg PO BID after euvolemic state has been achieved
 - Increase in furosemide dose to achieve euvolemia
 - Increase ACE inhibitor dose
 - Decrease ACE inhibitor dose

Explanation to above answer: The rhythm for question #16 above is complete heart block with obvious AV dissociation and therefore requires permanent pacemaker implantation.

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➤ Ventricular tachycardia

17. A 67-year-old woman has known coronary artery disease with a prior myocardial infarction. She presented with chest pain, dyspnea, and palpitations. She is in mild discomfort. Her blood pressure is 115/60. What is this rhythm?
- Ventricular tachycardia
 - Pre-excited supraventricular tachycardia (such as one with WPW may have)
 - Supraventricular tachycardia with aberrancy
 - None of the above
18. In the above patient's case (question #17), what is the most appropriate next step in management?
- Amiodarone, intravenously
 - Lidocaine, intravenously
 - Adenosine, intravenously
 - Direct current (DC) cardioversion
19. Corollary question to above case # 17:
- What would be the most appropriate management step if this patient's blood pressure was 80/40?
Answer: Direct current (DC) cardioversion, since this patient is now hemodynamically unstable.

References:

Edhouse J. Morris F. ABC of clinical electrocardiography: Broad complex tachycardia-Part I
BMJ. 324(7339):719-722, 2002 Mar 23.

Edhouse J. Morris F. ABC of clinical electrocardiography: Broad complex tachycardia-Part II.
BMJ. 324(7340):776-9, 2002 Mar 30.

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➤ Acute ST-elevation myocardial infarction

20. A 41-year-old man presented to the emergency department with severe chest pain, dyspnea, nausea, and diaphoresis—these symptoms began 2 hours ago while he was eating dinner. His past medical history is unremarkable. You obtain the following electrocardiogram. In addition to aspirin and intravenous heparin, what is the most appropriate next step in management?
- Administer intravenous glycoprotein IIb/IIIa inhibitor (e.g., eptifibatide) with heart catheterization planned for the morning or sooner if you cannot get him pain-free in 60-90 minutes
 - Administer intravenous thrombolytic therapy (e.g., tenecteplase)
 - Administer clopidogrel (Plavix) and intravenous nitroglycerin, followed by admission to the coronary care unit. This is electrocardiogram shows a left bundle branch block which precludes electrocardiographic diagnosis of acute myocardial injury
 - Insert a transvenous pacemaker immediately for 3rd degree atrioventricular block (complete heart block with AV dissociation)

Explanation to above answer: This tracing demonstrates an acute infero-lateral wall ST-elevation myocardial infarction with reciprocal ST segment depression; this condition requires immediate reperfusion therapy. The patient has an unremarkable past medical history and did not have any contraindications to thrombolytic therapy. Administering a glycoprotein IIb/IIIa inhibitor and waiting 60-90 minutes for pain to resolve is not appropriate. Primary PCI (percutaneous coronary intervention) would be the preferred reperfusion therapy (over thrombolytic therapy) if this patient had presented to a facility with capability for expert and prompt intervention (prompt = within 90 minutes of first medical contact).

References:

Morris F. Brady WJ. ABC of clinical electrocardiography: Acute myocardial infarction-Part I.
BMJ. 324(7341):831-4, 2002 Apr 6.

Edhouse J. Brady WJ. Morris F. ABC of clinical electrocardiography: Acute myocardial infarction-Part II. *BMJ*. 324(7343):963-6, 2002 Apr 20.

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➤ **Acute pericarditis**

21. A 68-year-old man who underwent surgical resection for esophageal cancer and now presents with severe chest pain. He appears acutely ill. His heart rate is 132/minute and blood pressure is 90/70. He has prominent elevation of his jugular venous pressure and is severely dyspneic and orthopneic. Which of the following is indicated next?
- Tenectapase (intravenous thrombolytic therapy), aspirin, and intravenous heparin
 - Enoxaparin in combination with eptifibatide prior to going emergently to the heart catheterization lab
 - Aspirin, clopidogrel (Plavix), intravenous heparin, and eptifibatide prior to going emergently to the heart catheterization lab
 - Aspirin, intravenous heparin, abciximab (Reopro) prior to going emergently to the cardiac catheterization lab
 - None of the above

Explanation to above answer: This particular patient had purulent pericarditis from his esophageal cancer resection. Any thrombolytic, anticoagulant, or anti-platelet therapy that can increase risk of bleeding (and therefore hemorrhagic tamponade) is strictly contraindicated.

Note the typical EKG features: depressed PR segment (or more generally, PR segment deviations opposite to P wave polarity), diffuse ST segment elevation, and sinus tachycardia.

References:

Imazio M, Trincheri R. Triage and management of acute pericarditis. *Int J Cardiol* 2007; 118:286.

Lange RA, Hillis LD. Clinical practice. Acute pericarditis. *N Engl J Med* 2004; 351:2195.

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➤ **Acute pulmonary thromboembolus with right ventricular strain pattern on EKG**

22. A 31-year-old woman with fibromyalgia developed sudden onset of chest pain which awakened her from sleep. She also developed concomitant dyspnea and appears very anxious when you meet her 45 minutes later in the emergency department. She is a non-smoker and does not take birth control pills. Of note, she severely sprained her ankle about 2 weeks ago and has been using crutches. Her heart rate is as shown, and her blood pressure is 80/60. What is the most likely diagnosis?
- Acute ST-elevation myocardial infarction
 - Acute pulmonary thromboembolism
 - Acute non-ST-elevation myocardial infarction
 - Cardiac tamponade from pericarditis
 - Anxiety/panic attack

23. Corollary questions to above case #22:

- a). What causes the classic S1, Q3, T3 pattern seen on EKG of a patient who presents with an acute pulmonary thromboembolus?

Answer: If the thromboembolus is large and causes obstruction of the pulmonary artery, then acute right ventricular strain with dilation will likely result which, in turn, causes a rightward shift of the QRS axis—manifesting as the S1, Q3, T3 pattern seen on EKG.

- b). If the thromboembolus is only a small one that occludes more distally in the pulmonary arterial tree (i.e., segmental or even subsegmental branch), what is a more common EKG finding in these cases?

Answer: Sinus tachycardia, or even a normal EKG.

References:

Harrigan RA. Jones K. ABC of clinical electrocardiography. Conditions affecting the right side of the heart. *BMJ*. 324(7347):1201-4, 2002 May 18.

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